

Effects of alterations in dietary carbohydrate intake on running performance during a 10 km treadmill time trial

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Abstract

Objective—To examine the influence of a seven day diet manipulation on performance during a 10 km treadmill time trial in trained runners.

Methods—Six trained runners ran two 10 km time trials on a treadmill set at a constant 4% gradient, each after a 7 d period of dietary manipulation. The two experimental diets were a low carbohydrate (CHO) diet (40% CHO by total energy) to be consumed for 7 d, and a high CHO diet containing 55% CHO for the first 4 d followed by 70% CHO for the remaining 3 d. Blood samples were obtained before and immediately after each run. Expired gases were collected and heart rate monitored.

Results—Performance time following the high CHO [48.8(SD 2.7) min] and low CHO [48.6(2.3) min] diets was not different ($P=0.72$), nor were there any differences in running speed between conditions. No significant differences were found between conditions in any of the metabolites measured (blood lactate, glucose, glycerol, and plasma free fatty acids). The rate of CHO oxidation was greater on the high CHO diet compared to the low CHO diet ($P<0.05$). Heart rate was not different between conditions.

Conclusions—The results of this study indicate that moderate changes in the composition of the diet do not affect 10 km running performance in trained subjects. (*Br J Sports Med* 1996;30:226-231)

Key terms: dietary manipulation; 10 km treadmill running performance; trained runners

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widespread use of pre-race CHO depletion/loading regimens among endurance athletes. However, in a later study involving well trained individuals, Sherman *et al*⁸ showed that, for individuals engaged in a daily programme of endurance training, the traditional CHO loading regimen did not cause muscle glycogen levels to be higher than did a more moderate programme involving a 50% CHO diet consumed for the first three days, followed by a high (70%) CHO diet consumed for the last three days before exercise. Similarly, Blom *et al*⁹ showed that it was possible for trained runners, performing regular training followed by two days with little or no running and a moderate CHO diet (400 g d⁻¹), to obtain muscle glycogen concentrations as high as those reported following the traditional CHO depletion/loading regimen.

While the early studies showed the importance of muscle glycogen in improving exercise capacity (time to fatigue at a fixed intensity), more attention is now directed towards the improvement of exercise performance (time to complete a set distance) by altering muscle glycogen concentration. However, only a limited number of studies have looked at the effects of altering muscle glycogen levels on running performance. In one of the earlier studies, Karlsson and Saltin¹⁰ showed that performance during a 30 km cross country race was improved following a CHO loading regimen. In that study, there was a considerable reduction in running speed in the later stages of the race when muscle glycogen approached low levels. Similarly, Williams *et al*¹¹ found that subjects ran faster during the last 5 km of a 30 km treadmill time trial after a seven day CHO supplemented diet compared to a normal mixed diet and concluded that extra dietary CHO improves running performance. On the other hand, over a shorter distance of 20.9 km, there was no improvement in running performance when subjects increased their muscle glycogen levels.⁸ These investigators concluded that increasing the muscle glycogen content above a certain value was not effective in improving performance.

The importance of increased pre-exercise muscle glycogen levels in improving performance during exercise lasting less than one hour has been questioned but has received little attention. The purpose of this study was to examine the influence of a seven day diet manipulation on performance during a 10 km treadmill time trial in trained runners.

The studies of Christensen and Hansen in the 1930s clearly showed the importance of carbohydrate (CHO) availability during exercise. This work was later expanded with the use of the percutaneous muscle biopsy technique to examine skeletal muscle glycogen metabolism during exercise in humans.¹² The ability to exercise for about 30 to 200 minutes seems to be limited mainly by the amount of glycogen stored in the working muscles before exercise.¹⁻⁵ High muscle glycogen stores have been shown to occur if muscles are depleted of their glycogen by prolonged exercise followed by a period of rest during which a high CHO diet is consumed.^{1,67} Such studies led to the

10 km Treadmill time trial (4% gradient)

Normal CHO diet (weighed intake)	Normal CHO diet	40% CHO diet						Normal CHO diet	40% CHO diet							
		OR							OR							
		55% CHO		70% CHO					55% CHO		70% CHO					
1 week	1 week	1	2	3	4	5	6	7	1	2	3	4	5	6	7	8

Figure 1 The experimental design. See text for details.

Methods

SUBJECTS

Six experienced male runners gave their written informed consent to take part in the present study which was approved by the local ethics committee. Their physical characteristics [mean(SD)] were: age 33(11) years; height 177(7) cm; body weight 71(5) kg; maximum oxygen consumption ($\dot{V}O_{2\max}$) 67(5) ml kg⁻¹ min⁻¹.

EXPERIMENTAL DESIGN

All subjects had their $\dot{V}O_{2\max}$ measured during an initial discontinuous incremental test on a motorised treadmill (Quinton Q65) and verified a few days later. The $\dot{V}O_{2\max}$ protocol required the subject to run in 3 min bouts, with 4–5 min rest periods between each bout, at a constant 4% gradient, while increasing the speed by 0.5 m s⁻¹ each time. During the last minute of each 3 min exercise bout expired air was collected and analysed using the Douglas bag method. From these results, the running speed equivalent to 80% of $\dot{V}O_{2\max}$ (at a constant 4% gradient) was determined for each subject. Subjects were then required to take part in at least two practice 10 km treadmill time trials in order to familiarise themselves with the test before undertaking the two experimental trials. A 4% gradient was used in order to reduce running speed and the impact on landing and therefore to limit the amount of muscle damage that tends to occur with treadmill running at high speeds. During all 10 km time trials, the treadmill speed was set at each subject's predetermined running speed for the first 2 km, after which subjects manually controlled the speed for the remainder of the run. Distance, speed, and time were printed out at 1 min intervals, using a treadmill logging program. This information was displayed on a monitor in front of the treadmill and acted as visual feedback for the subject.

Before the experimental trials, subjects were requested to follow their normal diet and weigh and record all food and drink consumed over a 7 d period. Normal training was continued and recorded during this period and was reproduced before each of the two experimental trials. The weighed dietary intake data were used to determine normal energy intake and diet composition using a computerised version of McCance and Widdowson's food composition tables, as revised by Holland *et al.*¹² These results were also used to design two diets to be consumed for the 7 d before the two experimental trials. The two experimental diets were a moderately low CHO diet (40% CHO) to be consumed for 7 d, and a high CHO diet

containing 55% CHO for the first 4 d followed by 70% CHO for the remaining 3 d. Both diets were isoenergetic with each subject's normal diet and their order was randomised. Subjects were given diet sheets containing the type, weight, and preparation of food and drink they were required to consume each day. Body weight measurements were obtained before and after the weighed intake and experimental diet periods, as well as before and after each treadmill time trial. The two experimental trials were separated by a period of two weeks in order to allow recovery from the previous performance trial. The experimental design is shown in fig 1.

PROCEDURES

Subjects reported to the laboratory in the morning 4 h after having consumed a standard breakfast [2.5(SD 1.4) MJ] with the same composition as the experimental diet. Once body weight was measured, subjects stood for 15 min with one hand immersed in water at 42°C in order to allow for arterialisation of the blood.¹³ Following this, a resting blood sample (5 ml) was obtained from an antecubital vein. A further blood sample was obtained immediately after completion of the run. Expired gas collections were obtained over a 60 s period at 1.5, 5.0, and 9.0 km for the determination of $\dot{V}O_2$ and respiratory exchange ratio (R). These values were further used to calculate the rate of CHO and fat oxidation for each time point. Heart rate was monitored at rest, at 0.5 km and at every km thereafter using a heart rate monitor (Polar PE 3000 Sport Tester). Room temperature was maintained between 22°C and 24°C.

BLOOD TREATMENT AND ANALYSIS

Blood (5 ml) was drawn into dry syringes and was dispensed equally into two tubes containing K₃EDTA. Duplicate aliquots (100 µl) from one of the K₃EDTA tubes were rapidly deproteinised in 1 ml of ice cold 0.3 mol litre⁻¹ perchloric acid; following centrifugation, the supernatant was used for the measurement of glycerol¹⁴ and glucose and lactate according to Maughan.¹⁵ A further aliquot of anticoagulated blood was centrifuged and the plasma obtained was separated and used for the measurement of free fatty acids (FFA) (colorimetric method, Boehringer Mannheim). The remainder of the blood was used for the measurement of haemoglobin (cyanmethaemoglobin method, Sigma) and packed cell volume (PCV, conventional microhaematocrit method). Changes in plasma volume were calculated from changes in haemoglobin and PCV relative to the pre-exercise values as described by Dill and Costill.¹⁶

DATA ANALYSIS

Statistical analysis of the data was carried out using two factor analysis of variance (ANOVA) for repeated measures followed by Student's *t* test for paired data, where necessary. All data are expressed as the mean(SD). Statistical significance was declared at *P* < 0.05.

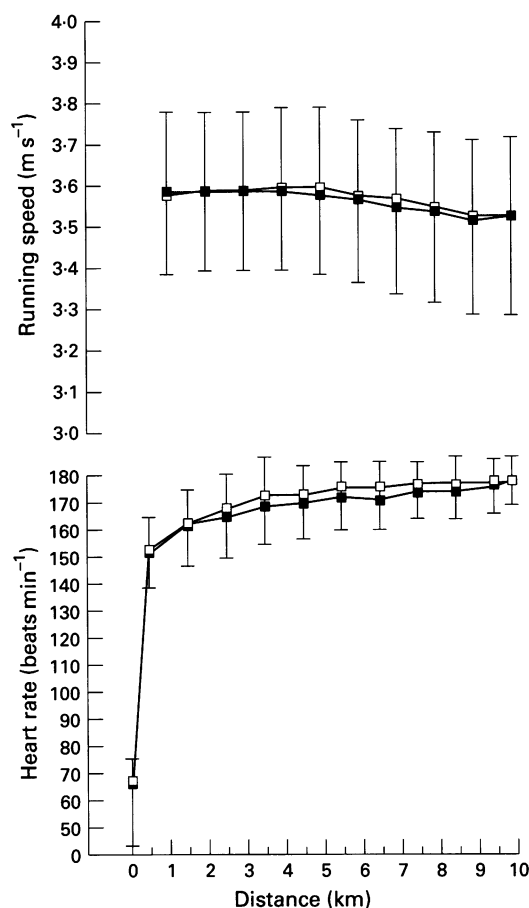


Figure 2 Running speed and heart rate, mean(SD), during the two performance trials following the high (■) and low (□) CHO diets ($n=6$).

Results

Mean performance time for the six subjects running on a treadmill set at a constant 4% gradient was not different following the high CHO [48.8(2.7)min] and low CHO [48.6(2.3)min] diets ($P=0.72$). Similarly, the running speed for each successive km of the 10 km treadmill time trial was the same between conditions (fig 2).

No differences were found between conditions in any of the metabolites measured (blood lactate, glucose, glycerol, and plasma free fatty acid)(table 1). Blood glucose, lactate, and glycerol increased during both trials. In contrast, plasma FFA concentration did not change during the run following the high CHO diet ($P=0.92$), while following the low CHO diet a reduction in FFA concentration was found ($P=0.03$). There was no change in plasma volume on either of the performance trials [0.1(5.5)% and -0.2(5.3)% following the high and low CHO diets, respectively].

The respiratory exchange ratio (R) tended to be higher during the run following the high CHO diet compared to the low CHO diet (fig 3) but no statistical difference was found ($P<0.10$). However, the rate of CHO oxidation was greater ($P<0.05$) on the high CHO diet compared to the low CHO diet [3.5(0.7) g min⁻¹ and 2.7(0.3) g min⁻¹, respectively]. A statistical difference was found at the 5.0 km time point ($P=0.01$). No significant difference in the rate of fat oxidation was found between

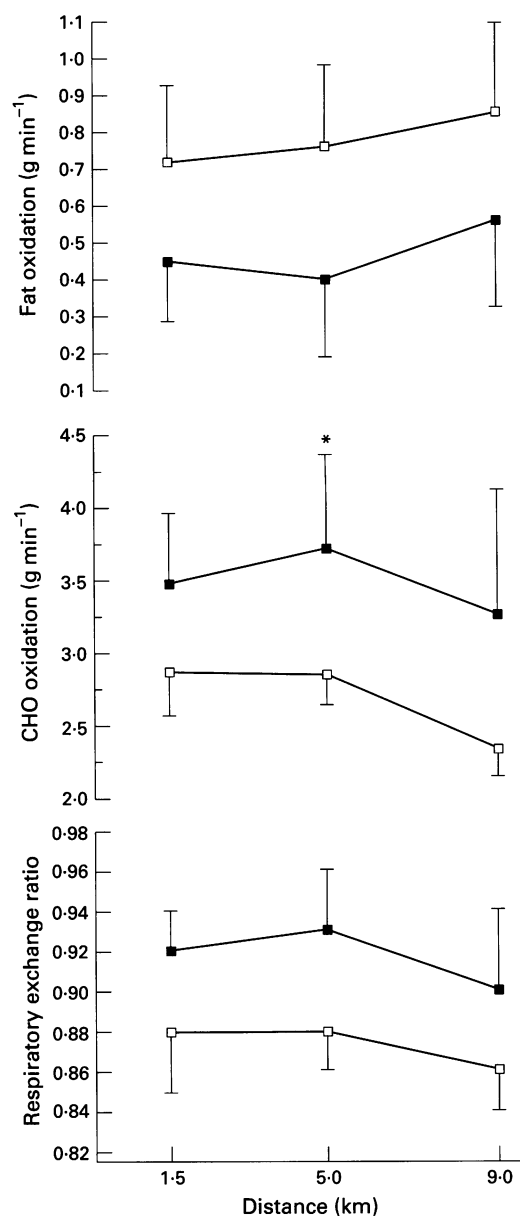


Figure 3 Respiratory exchange ratio and rates of CHO and fat oxidation, means(SD), during the two performance trials following the high (■) and low (□) CHO diets. *Significant difference ($P<0.05$) between conditions, $n=6$.

conditions despite the tendency ($P<0.10$) for the rate of fat oxidation to be lower during the high CHO trial (fig 3). At no time point during the entire run did the pre-exercise diet affect heart rate (fig 2) or $\dot{V}O_2$.

The habitual energy intake of the subjects was 13.7(3.2) MJ, of which 52(6)% of energy intake was in the form of CHO (table 2). Based on this energy intake, the two prescribed diets would have provided the subjects with approximately 350 g d⁻¹ of CHO during the low CHO diet compared to 482 g d⁻¹ (days 1-4) and 613 g d⁻¹ (days 5-7) of CHO during the high CHO diet. The subjects' normal training consisted of running 64(30) km week⁻¹ and was the same during the periods of dietary control.

No difference was found in body weight as a result of the two dietary conditions. However, a greater ($P=0.01$) weight loss occurred during the run following the low CHO diet [1.5(0.3) kg] compared to the high CHO diet [1.2(0.3) kg].

Table 1 Blood glucose, lactate, glycerol and plasma FFA concentrations, mean(SD), before and after the two performance trials (n=6).

	Glucose (mmol litre ⁻¹)		Lactate (mmol litre ⁻¹)		Glycerol (mmol litre ⁻¹)		FFA (mmol litre ⁻¹)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
High CHO diet	4.68 (0.74)	7.14* (0.93)	0.82 (0.16)	5.44* (1.41)	0.02 (0.02)	0.05* (0.02)	0.41 (0.32)	0.39 (0.28)
Low CHO diet	4.58 (0.58)	6.52* (1.12)	0.71 (0.20)	4.46* (1.63)	0.02 (0.02)	0.06* (0.05)	0.49 (0.28)	0.21* (0.11)

* P < 0.05 v pre-exercise values.

Table 2 Average daily energy intake and diet composition over the seven day weighed intake and the two dietary conditions. Values are presented as mean(SD).

	Normal	Low CHO	High CHO
Energy intake (MJ)	13.7 (3.2)	13.8 (3.3)	13.8 (3.2)
CHO (%)	52 (6)	40 (0)	55 (0)* 70 (0)†
Fat (%)	31 (5)	44 (3)	25 (3)
Protein (%)	13 (2)	14 (3)	12 (2)
Alcohol (%)	4 (6)	2 (3)	2 (3)

*(day 1-4).

†(day 5-7).

Subjects lost 2.2(0.3)% of body weight when running following the low CHO diet and 1.9(0.4)% of body weight following the high CHO diet (P<0.01). There was no difference in room temperature during the run following the high CHO diet and the low CHO diet: 23.2(0.7)°C v 23.3(0.8)°C.

Discussion

The results of this study indicate that moderate changes in the composition of the diet do not affect performance in an event in which the primary objective is to run a set distance of 10 km up a constant 4% gradient in as fast a time as possible. This finding is in agreement with the results of Sherman *et al.*⁸ who reported no improvements in running times in well trained runners during a 20.9 km run after a high CHO diet (70% CHO) despite higher pre-exercise muscle glycogen levels. Furthermore, in a more recent study, Sherman *et al.*¹⁷ showed that training on a low (5 g kg⁻¹ d⁻¹) CHO diet reduced total muscle glycogen but did not influence total time to exhaustion during two treadmill performance trials lasting approximately 10 minutes. These studies would suggest that the extra availability of substrate in the form of muscle glycogen does not improve performance or capacity for trained individuals in running events lasting less than 90 minutes.

Maximum running pace for an event of this distance is primarily dependent upon the rate of oxidation of both blood born fuels (FFA and glucose) and muscle glycogen: if muscle glycogen was reduced to such an extent that the blood born fuels could not provide energy at a sufficient rate to meet the demands of the exercising muscles, this would result in a reduction in running speed. The subjects in this study, however, were able to maintain the running speed set at the start on both trials, suggesting that adequate muscle glycogen remained in the exercising muscles at the end of the run. This finding is consistent with the observations of previous investigators who suggested that

differences in initial muscle glycogen stores would begin to influence maximum running pace only after muscle glycogen had approached low values (3-5 g kg⁻¹ wet weight).¹⁰ Similarly, Williams *et al.*¹¹ found that subjects ran faster only during the last 5 km of a 30 km treadmill time trial after a seven day CHO supplemented diet compared to a normal mixed diet. From these studies, it would seem that having a high initial level of muscle glycogen would allow the subject to maintain maximum running pace for longer, while having no effect on the maximum running speed the subject can maintain at the beginning of exercise.

The link between muscle glycogen depletion and fatigue during prolonged exercise was established in studies which involved exercise to exhaustion on a cycle ergometer.^{1-3 18} However, there appear to be different causes of fatigue during cycling versus running. Costill *et al.*¹⁹ showed that almost half of the initial glycogen remained within the quadriceps muscle after a 30 km run. Madsen *et al.*²⁰ found that some glycogen remained in both fibre types in the gastrocnemius muscle at the end of exhaustive treadmill running at 75-80% $\dot{V}O_2$ max lasting approximately 70-80 minutes. Even following a 42.2 km race completed in under three hours, Sherman *et al.*²¹ found glycogen concentrations well above zero (25 mmol kg⁻¹ wet weight) in the gastrocnemius muscle.

Because of the high energy intakes of these subjects, the low CHO diet continued to provide approximately 350 g of CHO per day. It may be argued that the amount of training carried out by the subjects during the period of study and the level of diet manipulation employed were not severe enough to affect running performance. If the amount of training was increased or the level of dietary CHO manipulation was more extreme to the extent that muscle glycogen was substantially reduced, running performance may well have been impaired. The analysis of the subjects' normal diet produced results similar to those previously reported by Williams *et al.*¹¹ who also used endurance trained club or recreational level runners.

Although muscle glycogen content was not determined in this study, the higher rate of CHO oxidation found during the high CHO trial suggests that the increased CHO intake had increased muscle glycogen. The mean total amount of CHO oxidised during the two performance trials was 171 g and 131 g for the high and low CHO trials, respectively. However, since no difference in performance was found between conditions, this higher rate of

CHO oxidation on the high CHO trial would suggest that CHO availability was not limiting running performance in this study. This finding is consistent with the study by Sherman *et al.*,⁸ who found a greater CHO utilisation but no improvements in running performance. Similarly, in the study by Williams *et al.*,¹¹ 33 g more CHO was utilised during a 30 km treadmill time trial following a seven day high CHO diet than with a control diet, but no improvement in overall running performance was found.

It is well established that feeding diets very high in CHO (>60%) will result in higher blood lactate levels compared to diets very low (<12%) in CHO.¹²⁻²⁷ This effect of diet on blood lactate may be the result of increased glycogen levels, as Richter and Galbo²⁸ have shown that increased concentrations of glycogen in isolated muscle led to an increased breakdown of glycogen and release of lactate during muscle contraction. This effect of diet on blood lactate concentration has also been attributed to changes in acid-base status,²⁹ probably as a direct consequence of variations in protein intake.^{30,31} In the present study, although blood lactate concentration following the high CHO diet tended to be higher than on the low CHO diet, this difference was not statistically different. This lack of effect of diet on blood lactate is probably the result of the moderate nature of the current diet manipulation, even though the rate of CHO utilisation was increased following the high CHO diet. During 25 minutes of exercise at 65-70% $\text{VO}_{2\text{max}}$ following extreme dietary conditions (5% and 75% CHO diets), Jansson³² showed that R agreed closely with the respiratory quotient measured directly across the working muscles, therefore allowing CHO utilisation in skeletal muscle to be determined using R even under conditions of altered blood lactate. Using the same protocol, Jansson and Kaijser³³ also found a higher R and therefore greater CHO utilisation associated with a greater muscle lactate accumulation and release following the 75% CHO diet compared to the 5% CHO diet. These workers attributed the greater lactate concentrations found during this condition to a higher rate of muscle glycogenolysis.

Hultman and Nilsson³⁴ showed that when dietary CHO intake is substantially reduced, liver glycogen stores decrease rapidly and they concluded that under such circumstances liver glycogen stores are unable to meet the demands of heavy exercise for glucose and a drop in blood glucose results. In the present study, however, blood glucose rose during the run on both trials and was not different between conditions. This observation would suggest an adequate release of glucose from the liver for peripheral utilisation despite any alterations in liver glycogen as a result of the dietary manipulation. The similar rise in blood glucose during both conditions is further evidence that CHO availability was not limiting running performance and that the hyperglycaemia associated with very high intensity exercise^{25,35,36} can also be elicited with the lower intensity treadmill running protocol used in

the present study following both a high and low CHO diet.

While no difference in plasma FFA concentration was found between conditions, a reduction in FFA concentration was found at the end of the run following the low CHO diet: no difference was found following the high CHO diet. This observation would suggest that during the run following the high CHO diet the mobilisation and transport of FFA into the blood was sufficient to supply a larger fraction of the total fuel than was actually used and that these processes were not limiting. On the other hand, the reduction in plasma FFA concentration observed during the low CHO trial would point towards an increase in energy contribution from plasma FFA in order to sustain the running speed set at the start. While the tendency was for the rate of fat oxidation to be higher during the low CHO trial, no statistical difference was found. It is not possible from the present results to determine whether the reduction in plasma FFA concentration found at the end of the run following the low CHO diet indicates that there was an increase in re-esterification of FFA during this trial. Differences in mobilisation of FFA during the two trials can, however, be excluded as FFA and glycerol concentrations did not vary between conditions.

The increase in blood glycerol at the end of both trials and the finding of similar concentrations between conditions is in agreement with the results reported in the previous performance study by Williams *et al.*¹¹ However, only a threefold increase in plasma glycerol concentration was observed in the present study compared to almost a 10-fold increase in the study by Williams and co-workers. This difference is probably the result of the greater mobilisation of fat evident from the much higher plasma FFA concentrations reported by these investigators at the end of the 30 km treadmill time trials.

The fact that the subjects involved in this study were all endurance trained may have contributed to the lack of effect of diet on running performance. A major effect of endurance training is to increase the utilisation of fat while at the same time reducing the utilisation of CHO during submaximal exercise.³⁷ The proportions of CHO and fat oxidised during exercise are also dependent on the availability of substrate. During the low CHO trial, therefore, the endurance trained status of these subjects may have allowed for a larger proportion of the energy required to maintain the subjects' maximum running pace to be provided by the oxidation of fat compared to the high CHO trial. Had we used untrained subjects in this study, their lower ability to utilise fat may have led to slower running times following the low CHO diet.

Weight loss during the performance trials was different. Subjects lost approximately 1.9% body weight on the high CHO diet and 2.2% body weight on the low CHO diet. Since subjects did not drink during the performance trials, and there was no difference in room temperature between conditions, this observa-

tion would suggest that subjects sweated more on the low CHO diet compared to the high CHO diet. However, there seems to be no reasonable explanation for this finding, and as the differences in body weight are small they probably occurred by chance.

In summary, these results indicate that moderate changes in the composition of the diet do not affect 10 km running performance in endurance trained subjects. However, the higher rate of CHO oxidation found during the high CHO trial suggests that the increased CHO intake had increased muscle glycogen. However, since no difference in performance was found between conditions, this higher rate of CHO oxidation on the high CHO trial would suggest that CHO availability was not limiting running performance in this study.

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